Myers
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21feb02 10:22:24 User219783 Session D1795.6

SYSTEM:OS - [redacted]
File 440:Current Contents Search(R) 1990-2002/Feb W4
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File 149:TGG Health&Wellness DB(SM) 1976-2002/Feb W2
(c) 2002 The Gale Group
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File 144:Pascal 1973-2002/Feb W3
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File 484:Periodical Abs Plustext 1986-2002/Feb W3
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File 98:General Sci Abs/Full-Text 1984-2002/Jan
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File 50:CAB Abstracts 1972-2002/Jan
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File 370:Science 1996-1999/Jul W3
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*File 370: This file is closed (no updates). Use File 47 for more current
information.
File 44:Aquatic Sci&Fish Abs 1978-2002/Jan

Searcher : Shears 308-4994

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  File 442:AMA  Journals  1982-2002/Mar B1
        (c)2002 Amer Med Assn -FARS/DARS apply
*File 442: UDs have been adjusted to reflect the current months
data. See Help News442 for details. PY,PD sort temporarily do not work.
  File 444:New England Journal of Med.  1985-2002/Feb W4
        (c) 2002 Mass. Med. Soc.
  File  10:AGRICOLA  70-2002/Feb
        (c) format only 2002 The Dialog Corporation
  File  16:Gale Group PROMT(R)  1990-2002/Feb 20
        (c) 2002 The Gale Group
  File  65:Inside Conferences  1993-2002/Feb W3
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  File  99:Wilson Appl. Sci & Tech Abs  1983-2002/Jan
        (c) 2002 The HW Wilson Co.
  File 135:NewsRx Weekly Reports  1995-2002/Feb W3
        (c) 2002 NewsRx
  File 162:CAB HEALTH  1983-2002/Jan
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*File 162: Truncating CC codes is recommended for full retrieval.
See Help News162 for details.
  File 172:EMBASE Alert  2002/Feb W3
        (c) 2002 Elsevier Science B.V.
  File 185:Zoological Record Online(R)  1978-2001/Dec
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  File 453:Drugs of the Future  1990-2001/Dec
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  File 636:Gale Group Newsletter DB(TM)  1987-2002/Feb 20
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      Set  Items  Description
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Set     Items   Description
S1       4908   TCFI OR TCF1 OR (TCF OR T(W)CELL(1W)TRANSCRIPT?(W)FACTOR)(-
                  2N)(1 OR I)
S2        633   S1 AND (POLYMORPH? OR POLY(W)(MORPHIS? OR MORPHIC?) OR MUT-
                  AGEN? OR MUTAT? OR MUTANT? ? OR ALLEL? OR VARIANT? ?)


S5        545   S2 AND GENE? ?


S14       533   S1(10N)GENE? ?
S15        80   S14(10N)(POLYMORPH? OR POLY(W)(MORPHIS? OR MORPHIC?) OR MU-
                  TAGEN? OR MUTAT? OR MUTANT? ? OR ALLEL? OR VARIANT? ?)

S17        64   S5 AND (C883 OR CYS883 OR (C OR CYSTEIN? OR CYS)(W)883 OR -
                  EXON? ?)
S18         1   S5 AND (POSITION OR RESIDUE)(W)883
S19       121   S15 OR S17 OR S18
S20        44   RD (unique items)
>>>No matching display code(s) found in file(s): 65, 135, 453

 20/3,AB/1     (Item 1 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)
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13097504   GENUINE ARTICLE#: 477QF   NUMBER OF REFERENCES: 19
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-Key terms

TITLE: Analysis of a non-functional HNF-1 alpha (TCF1) mutation in Japanese subjects with familial type 1 diabetes
AUTHOR(S): Yoshiuchi I; Yamagata K (REPRINT); Yoshimoto M; Zhu Q; Yang Q; Nammo T; Uenaka R; Kinoshita E; Hanafusa T; Miyagawa J; Matsuzawa Y
AUTHOR(S) E-MAIL: kazu@imed2.med.osaka-u.ac.jp
CORPORATE SOURCE: Osaka Univ, Dept Internal Med & Mol Sci, B5,2-2 Yamadaoka/Suita/Osaka 5650871/Japan/ (REPRINT); Osaka Univ, Dept Internal Med & Mol Sci, /Suita/Osaka 5650871/Japan/; Childrens Clin Yoshimoto, /Nagasaki//Japan/; Nagasaki Univ, Dept Pediat, /Nagasaki 852//Japan/; Osaka Med Coll, Dept Internal Med 1, /Osaka//Japan/
PUBLICATION TYPE: JOURNAL
PUBLICATION: HUMAN MUTATION, 2001, V18, N4, P345-351
PUBLISHER: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012 USA
ISSN: 1059-7794
LANGUAGE: English DOCUMENT TYPE: ARTICLE
ABSTRACT: Mutations in the transcription factor hepatocyte nuclear factor-1 alpha (HNF-1 alpha; gene symbol TCF1) cause maturity onset diabetes of the young type 3 (MODY3), a form of diabetes mellitus characterized by autosomal dominant inheritance, early onset, and pancreatic beta -cell dysfunction. Recent genetic studies, however, also found mutations in patients diagnosed with idiopathic (non autoimmune based) type 1 diabetes. We identified a novel frameshift *mutation"** (142delG) in the *TCF1"** *gene"** in a family with a strong family history of type I diabetes and examined the functional proper ties of the mutant HNF 1 alpha The expression of the mutant protein was not detected in COS-7 cells by Western blot analysis after transfection of the mutant cDNA. This is the first case of an unstable mutant HNF-1 alpha protein. Reporter gene analysis indicated that the mutant HNF-1 alpha had no transactivation activity in HeLa and MIN6 cells. Haploinsufficiency for HNF-1 alpha may lead to severe forms of diabetes like type 1 diabetes. Hum Mutat 18:345-351, 2001. (C) 2001 Wiley-Liss, Inc.

20/3,AB/2 (Item 2 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

13042180 GENUINE ARTICLE#: 469AG NUMBER OF REFERENCES: 30
TITLE: Genetic alterations in the human Tcf-4 *gene"** in Japanese patients with sporadic gastrointestinal cancers with microsatellite instability
AUTHOR(S): Saeki H (REPRINT); Tanaka S; Tokunaga E; Kawaguchi H; Ikeda Y; Maehara Y; Sugimachi K
AUTHOR(S) E-MAIL: hsaeki@med.kyushu-u.ac.jp
CORPORATE SOURCE: Kyushu Univ, Higashi Ku, 3-1-1 Maidashi/Fukuoka 8128582//Japan/ (REPRINT); Kyushu Univ, Higashi Ku, /Fukuoka 8128582//Japan/
PUBLICATION TYPE: JOURNAL
PUBLICATION: ONCOLOGY, 2001, V61, N2, P156-161
PUBLISHER: KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND
ISSN: 0030-2414
LANGUAGE: English DOCUMENT TYPE: ARTICLE
ABSTRACT: Disruption of the APC/beta -catenin/Tcf pathway has been proposed as an important step in the development of cancer. The Tcf-4 transcription factor *gene"** was reported to be one of the targets of microsatellite instability (MSI) in colorectal cancers in with MSI. We carried out a sequencing analysis of the Tcf-4 *gene"** in 41 Japanese patients with gastrointestinal tumors with MSI as well as in cancer

cell lines. Three of 21 (14.3%) colorectal tumors with MSI contained a *mutant"** Tcf-4 *gene"** encoding 1-bp deletion in an (A)9 repeat, leading to carboxyl terminal truncation of Tcf-4 protein. No Tcf-4 *mutations"** were detected in 20 gastric tumors with MSI, or in gastric cancer cell lines. Additionally, we found a novel transcript of the Tcf-4 *gene"** which contained a segment of 73 bp in front of the (A)9 repeat of the Tcf-4 coding sequence. Sequencing analysis revealed that the inserted fragment was 60% homologous to that of *exon"** IXA of the *Tcf"**-*1"** *gene"**. It is of interest that this insertion resulted intruncation of Tcf-4, similar to the 1-bp deletion in the (A)9 repeat. Therefore, in part of the Japanese colorectal tumors with MSI, frameshift *mutations"** in Tcf-4 may be of functional significance. Functional alterations in the Tcf-4 signaling network in gastrointestinal tumorigenesis require further investigations.

20/3,AB/3 (Item 3 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

12906105 GENUINE ARTICLE#: 454VY NUMBER OF REFERENCES: 20
TITLE: *Mutations"** of the beta- and gamma-catenin *genes"** are uncommon in human lung, breast, kidney, cervical and ovarian carcinomas
AUTHOR(S): Ueda M (REPRINT); Gemmill R; West J; Winn R; Sugita M; Tanaka N; Ueki M; Drabkin HA
CORPORATE SOURCE: Osaka Med Coll, Dept Obstet & Gynecol, 2-7 Daigakumachi/Osaka 5698686//Japan/ (REPRINT); Osaka Med Coll, Dept Obstet & Gynecol, /Osaka 5698686//Japan/; Univ Colorado, Div Med Oncol, /Denver//CO/80262; Univ Colorado, Dept Pathol, /Denver//CO/80262; Chiba Univ, Chuo Ku, /Chiba 2608670//Japan/
PUBLICATION TYPE: JOURNAL
PUBLICATION: BRITISH JOURNAL OF CANCER, 2001, V85, N1 (JUL 6), P64-68
PUBLISHER: CHURCHILL LIVINGSTONE, JOURNAL PRODUCTION DEPT, ROBERT STEVENSON HOUSE, 1-3 BAXTERS PLACE, LEITH WALK, EDINBURGH EH1 3AF, MIDLOTHIAN, SCOTLAND
ISSN: 0007-0920
LANGUAGE: English DOCUMENT TYPE: ARTICLE
ABSTRACT: beta -catenin forms complexes with *Tcf"** and Lef-*1"** and functions as a transcriptional activator in the Wnt signalling pathway. Although recent investigations have been focused on the role of the adenomatous polyposis coli (APC)/beta -catenin/Tcf pathway in human tumorigenesis, there have been very few reports on *mutations"** of the beta -catenin *gene"** in a variety of tumour types. Using PCR and single-strand conformational *polymorphism"** analysis. we examined 93 lung, 9 breast, 6 kidney, 19 cervical and 7 ovarian carcinoma cell lines for *mutations"** in *exon"** 3 of the beta -catenin *gene"**. In addition, we tested these same samples for *mutations"** in the NH2-terminal regulatory region of the gamma -catenin *gene"**. *Mutational"** analysis for the entire coding region of beta -catenin cDNA was also undertaken in 20 lung. 9 breast. 5 kidney and 6 cervical carcinoma cell lines. Deletion of most beta -catenin coding *exons"** was confirmed in line NCl-H28 (lung mesothelioma) and a silent *mutation"** at codon 214 in *exon"** 5 was found in HeLa (cervical adenocarcinoma). A missense *mutation"** at codon 19 and a silent *mutation"** at codon 28 in the NH2-terminal regulatory region of the gamma -catenin *gene"** were found in H1726 (squamous cell lung carcinoma) and H1048 (small cell lung carcinoma), respectively. Neither

deletions nor *mutations"** of these *genes"** were detected in the other cell lines examined. These results suggest that beta- and gamma -catenins are infrequent *mutational"** targets during development of human lung, breast, kidney, cervical and ovarian carcinomas. (C) 2001 Cancer Research Campaign http://www.bjcancer.com.

20/3,AB/4 (Item 4 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

12899424 GENUINE ARTICLE#: 454HK NUMBER OF REFERENCES: 45
TITLE: Rescue of a Wnt mutation by an activated form of LEF-1: Regulation of maintenance but not initiation of Brachyury expression
AUTHOR(S): Galceran J; Hsu SC; Grosschedl R (REPRINT)
AUTHOR(S) E-MAIL: rgross@lmb.uni-muenchen.de
CORPORATE SOURCE: Univ Munich, Gene Ctr, Feodor Lynenstr 25/D-81377 Munich//Germany/ (REPRINT); Univ Munich, Gene Ctr, /D-81377 Munich//Germany/; Univ Munich, Inst Biochem, /D-81377 Munich//Germany/
PUBLICATION TYPE: JOURNAL
PUBLICATION: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, 2001, V98, N15 (JUL 17), P8668-8673
PUBLISHER: NATL ACAD SCIENCES, 2101 CONSTITUTION AVE NW, WASHINGTON, DC 20418 USA
ISSN: 0027-8424
LANGUAGE: English DOCUMENT TYPE: ARTICLE
ABSTRACT: Members of the LEF-1/TCF family of transcription factors have been implicated in mediating a nuclear response to Wnt signals by association with beta -catenin, consistent with this view, mice carrying *mutations"** in either the Wnt3a *gene"** or in both transcription factor *genes"** Lef1 and *Tcf1"** were previously found to show a similar defect in the formation of paraxial mesoderm in the gastrulating mouse embryo. In addition, mutations in the Brachyury gene, a direct transcriptional target of LEF-1, were shown to result in mesodermal defects. However, direct evidence for the role of LEF-1 and Brachyury in Wnt3a signaling has been limiting. In this study, we genetically examine the function of LEF-1 in the regulation of Brachyury expression and in signaling by Wnt3a. Analysis of the expression of Brachyury in Lef1(-/-)Tcf1(-/-) mice and studies of Brachyury:lacZ transgenes containing wild type or mutated LEF-1 binding sites indicate that Lef1 is dispensable for the initiation, but is required for the maintenance of Brachyury expression. We also show that the expression of an activated form of LEF-1, containing the beta -catenin activation domain fused to the amino terminus of LEF-1, can rescue a Wnt3a mutation. Together, these data provide genetic evidence that Lef1 mediates the Wnt3a signal and regulates the stable maintenance of Brachyury expression during gastrulation.

20/3,AB/5 (Item 5 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

12481005 GENUINE ARTICLE#: 407KR NUMBER OF REFERENCES: 38
TITLE: Role of common sequence *variants"** in insulin secretion in familial type 2 diabetic kindreds - The sulfonylurea 1-receptor, glucokinase, and hepatocyte nuclear factor 1 alpha *genes"**
AUTHOR(S): Elbein SC (REPRINT); Sun JP; Scroggin E; Teng K; Hasstedt SJ

AUTHOR(S) E-MAIL: elbeinstevenc@exchange.uams.edu
CORPORATE SOURCE: John L McClellan Mem Vet Hosp, 111J-LR,4300 W 7th St/Little Rock//AR/72205 (REPRINT); Cent Arkansas Vet Healthcare Syst, Div Endocrinol, /Little Rock//AR/; Univ Arkansas Med Sci, /Little Rock//AR/72205; Univ Utah, Dept Human Genet, /Salt Lake City//UT/84132
PUBLICATION TYPE: JOURNAL
PUBLICATION: DIABETES CARE, 2001, V24, N3 (MAR), P472-478
PUBLISHER: AMER DIABETES ASSOC, 1660 DUKE ST, ALEXANDRIA, VA 22314 USA
ISSN: 0149-5992
LANGUAGE: English DOCUMENT TYPE: ARTICLE
ABSTRACT: OBJECTIVE - We have demonstrated high heritability of insulin secretion measured as acute insulin response to glucose times insulin sensitivity (disposition index). Furthermore, me showed that obese normoglycemic family members of a type 2 diabetic proband Failed to compensate for the insulin resistance for obesity by increasing insulin secretion. In this study, we tested the primary hypotheses that previously described *variants"** in the pancreatic sulfonylurea receptor *gene"** (SUR1 or ABCC8), glucokinase (GCK) gent, or hepatocyte nuclear factor 1 alpha (*TCF1"** or HNF1 alpha) *gene"** contribute to the inherited deficiencies of insulin secretion and beta -cell compensation to insulin resistance, as well as the secondary hypotheses that these *variants"** altered insulin sensitivity.

RESEARCH DESIGN AND METHODS - We typed 124 nondiabetic member 5 of 26 familial type 2 diabetic kindreds who had undergone tolbutamide-modified intravenous glucose tolerance tests for two *variants"** of the ABCC8 (sulfonylurea) *gene"**, two *variants"** of the GCK *gene"**, and one common amino acid *variant"** in the *TCF1"** (HNF1 alpha) *gene"**. All family members were classified as normal or having impaired glucose tolerance based on oral glucose tolerance testing. We used minimal model analysis to calculate the insulin sensitivity index (S-1) and glucose effectiveness (S-G), and acute insulin response to glucose was calculated as the mean insulin excursion above baseline during the first 10 min after the glucose bolus. Disposition index (DI), a measure of beta -cell compensation for insulin sensitivity, was calculated as insulin sensitivity times acute insulin response. Effects of *polymorphisms"** were determined using mixed effects models that incorporated family membership and by a likelihood analysis that accounted for family structure through polygenic inheritance.

RESULTS - An intronic *variant"** of the ABCC8 *gene"** just upstream of *exon"** 16 was a significant determinant of both DI and an analogous index based on acute insulin response to tolbutamide. Surprisingly, heterozygous individuals showed the lowest indexes, whereas the DI in the two homozygous states did not differ significantly. Neither the *exon"** 18 *variant"** nor the *variants"** in the GCK and *TCF1"** *genes"** were significant in this model. However, combined genotypes of ABCC8 *exon"** 16 and 18 *variants"** again significantly predicted both indexes of glucose and tolbutamide-stimulated insulin secretion. Unexpectedly, a *variant"** in the 3' untranslated region of the GCK *gene"** interacted significantly with BMI to predict insulin sensitivity.

CONCLUSIONS - The *exon"** 16 *variant"** of the ABCC8 *gene"** reduced beta -cell compensation to the decreased insulin sensitivity in the heterozygous state. This may explain the observation from several groups of an association of the ABCC8 *variants"** in diabetes and is

consistent with other studies showing a role of ABCC8 *variants"** in pancreatic beta -cell function. However, our study focused on individuals from relatively few families, Ascertainment bias, family structure, and other interacting *genes"** might have: influenced our unexpected result, Additional studies are needed to replicate our observed deficit in beta -cell compensation in individuals heterozygous for ABCC8 *variants"**. Likewise, the role of the GCK 3' *variant"** in the reduced insulin sensitivity of obesity will require further study.

20/3,AB/6 (Item 6 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

12127338 GENUINE ARTICLE#: 368KH NUMBER OF REFERENCES: 59
TITLE: Hepatocyte nuclear factor 1 alpha (HNF-1 alpha) *mutations"** in maturity-onset diabetes of the young
AUTHOR(S): Ellard S (REPRINT)
AUTHOR(S) E-MAIL: S.Ellard@exeter.ac.uk
CORPORATE SOURCE: Royal Devon & Exeter NHS Healthcare Trust, Genet Mol Lab, Barrack Rd/Exeter EX2 5DW/Devon/England/ (REPRINT); Univ Exeter, Dept Vasc Med & Diabet Res, /Exeter/Devon/England/; Royal Devon & Exeter NHS Healthcare Trust, Genet Mol Lab, /Exeter EX2 5DW/Devon/England/
PUBLICATION TYPE: JOURNAL
PUBLICATION: HUMAN MUTATION, 2000, V16, N5, P377-385
PUBLISHER: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012 USA
ISSN: 1059-7794
LANGUAGE: English DOCUMENT TYPE: ARTICLE
ABSTRACT: Maturity-onset diabetes of the young (MODY) is a monogenic form of diabetes mellitus characterized by autosomal dominant inheritance, early age of onset (< 25 years) and pancreatic <beta>-cell dysfunction. MODY is genetically heterogeneous with five different *genes"** identified to date: hepatocyte nuclear factor-4 alpha (HNF-4 alpha) [MODY1]; glucokinase [MODY2]; hepatocyte nuclear factor-1 alpha (HNF-1 alpha) [MODY3]; insulin promoter factor-1 (IPF-1) [MODY4]; and hepatocyte nuclear factor-1 beta (HNF-1 beta) [MODY5], *Mutations"** in the HNF-1 alpha *gene"** represent a common cause of MODY in the majority of populations studied. Sixty-five different *mutations"** have been described in a total of 116 families. The most common *mutation"** is a C-insertion (P291fsinsC) in the polyC tract of *exon"** 4, which has been reported in 22 families. The identification of an HNF-1 alpha *gene"** *mutation"** in a patient with type 2 diabetes confirms the diagnosis of MODY and has important implications for clinical management. Hum *Mutat"** 16:377-385, 2000, (C) 2000 Wiley-Liss, Inc.

20/3,AB/7 (Item 7 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

11299485 GENUINE ARTICLE#: 278BV NUMBER OF REFERENCES: 6
TITLE: Big Dye (TM) Terminator Cycle Sequencing Chemistry: Accuracy of the dilution process and application for screening *mutations"** in the *TCF1"** and GCK *genes"**
AUTHOR(S): Boutin P (REPRINT); Wahl C; Samson C; Vasseur F; Laget F; Froguel P

AUTHOR(S) E-MAIL: philippe.boutin@pasteur-lille.fr
CORPORATE SOURCE: Inst Pasteur, CNRS,Dept Human Genet, 1 Rue du Pr Calmette/F-59019 Lille//France/ (REPRINT); Inst Pasteur, CNRS,Dept Human Genet, /F-59019 Lille//France/; PE Biosyst, /Courtaboeuf//France/
PUBLICATION TYPE: JOURNAL
PUBLICATION: HUMAN MUTATION, 2000, V15, N2, P201-203
PUBLISHER: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012 USA
ISSN: 1059-7794
LANGUAGE: English DOCUMENT TYPE: LETTER

20/3,AB/8 (Item 8 from file: 440)

10000601 GENUINE ARTICLE#: 137BG NUMBER OF REFERENCES: 18
TITLE: Hepatocyte nuclear factor-1 alpha gene and non insulin dependent diabetes mellitus in the Japanese population
AUTHOR(S): Babaya N; Ikegami H (REPRINT); Kawaguchi Y; Fujisawa T; Nakagawa Y; Hamada Y; Hotta M; Ueda H; Shintani M; Nojima K; Kawabata E; Ono M; Yamada K; Shen GQ; Fukuda M; Ogihara T
CORPORATE SOURCE: OSAKA UNIV,SCH MED, DEPT GERIATR MED, 2-2 YAMADAOKA/SUITA/OSAKA 565/JAPAN/ (REPRINT); OSAKA UNIV,SCH MED, DEPT GERIATR MED/SUITA/OSAKA 565/JAPAN/; OSAKA TEISHIN HOSP,/OSAKA//JAPAN/
PUBLICATION TYPE: JOURNAL
PUBLICATION: ACTA DIABETOLOGICA, 1998, V35, N3, P150-153
PUBLISHER: SPRINGER VERLAG, 175 FIFTH AVE, NEW YORK, NY 10010
ISSN: 0940-5429
LANGUAGE: English DOCUMENT TYPE: ARTICLE
ABSTRACT: Recently, hepatocyte nuclear factor-1 alpha (HNF-1 alpha which is encoded by the *TCF1"** *gene"**) *mutations"** were reported in a subset of patients with maturity onset diabetes of the young (MODY3). We studied the contribution of TCF1 to genetic susceptibility to common non-insulin-dependent diabetes mellitus (type 2) in Japanese subjects by investigating allelic association with type 2 diabetes use of three markers. We also studied the frequency of the G191D mutation, the only mutation of TCF1 reported so far in late-onset type 2 diabetes. A total of 356 subjects were studied. There were no significant differences in allele frequency of the three markers between patients with type 2 diabetes and control subjects. A G191D mutation was not found in the subjects studied, giving a frequency of less than 0.4% in common type 2 diabetes. The lack of association of type 2 diabetes with three markers in and near TCF1 suggests that mutations in TCF1 derived from a limited number of founders are not a major cause of common type 2 diabetes even in the genetically homogeneous Japanese population. The data also indicate that the G191D mutation in TCF1 plays little, if any, role in susceptibility to common type 2 diabetes in the Japanese.

20/3,AB/9 (Item 9 from file: 440)

09921307 GENUINE ARTICLE#: 128TA NUMBER OF REFERENCES: 13
TITLE: Predictive value of nuclear beta-catenin expression for the occurrence of distant metastases in rectal cancer
AUTHOR(S): Gunther K (REPRINT); Brabletz T; Kraus C; Dworak O; Reymond MA;

Jung A; Hohenberger W; Kirchner T; Kockerling F; Ballhausen WG
CORPORATE SOURCE: UNIV ERLANGEN NURNBERG,CHIRURG KLIN & POLIKLIN, KRANKENHAUSSTR 12/D-91054 ERLANGEN//GERMANY/ (REPRINT); UNIV ERLANGEN NURNBERG,DEPT SURG/D-8520 ERLANGEN//GERMANY/; UNIV ERLANGEN NURNBERG,DEPT PATHOL/D-8520 ERLANGEN//GERMANY/; UNIV ERLANGEN NURNBERG,INST HUMAN GENET/D-8520 ERLANGEN//GERMANY/
PUBLICATION TYPE: JOURNAL
PUBLICATION: DISEASES OF THE COLON & RECTUM, 1998, V41, N10 (OCT), P 1256-1261
PUBLISHER: WILLIAMS & WILKINS, 351 WEST CAMDEN ST, BALTIMORE, MD 21201-2436
ISSN: 0012-3706
LANGUAGE: English DOCUMENT TYPE: ARTICLE
ABSTRACT: Adenomatous polyposis coli protein, glycogen synthetase kinase-3-beta, T cell transcription factor/lymphoid enhancer-binding factor, and beta-catenin modulate cell differentiation and proliferation via the expression of effector *genes"**. It has recently been postulated that beta-catenin isa potent oncogene of sporadic colorectal carcinogenesis and a prognostic tumor marker. Our aim was to investigate whether the nuclear overexpression of beta-catenin, possibly caused by *mutations"** in *exon"** 3 of beta-catenin (CTNNB1), is correlated with distant metastatic spread or disease-free survival in rectal carcinoma. METHODS: Immunohistochemical analysis was performed with an anti-beta-catenin-monoclonal antibody on paraffin sections of two groups of patients (n = 2 X 77) with rectal carcinoma curatively treated by surgery alone. The patients selected were all free of local disease, to exclude surgical influence. Patient groups were matched for age, gender, International Union Against Cancer stage, and year of operation (1982 to 1991) and differed only in subsequent metachronous distant metastatic spread. Follow-up was prospective (median, 9.6 years). Three staining patterns were defined: membranous (normal), diffuse cytoplasmic (pathologic), and intense nuclear staining (pathologic). When intense nuclear staining was defined, the specimen was microdissected. Then, DNA was isolated, polymerase chain reaction-amplified, and sequenced to detect *mutations"** in *exon"** 3. RESULTS: Nuclear overexpression of beta-catenin correlated neither with distant metastatic spread (chi-squared, 0.37; P = 0.79) nor with disease-free survival (log-rank: with trend, P = 0.62). No *mutations"** were found in the area of the serine/threonine-kinase glycogen synthetase kinase-3-beta-phosphorylation site in *exon"** 3 (CTNNB1) of beta-catenin. CONCLUSION: Although beta-catenin seems to play an important rule in early colorectal carcinogenesis, its value as a prognostic marker is questionable. It must be: assumed that metastatic ability is determined by other factors than the disturbance of the beta-catenin T cell transcription factor/lymphoid enhancer-binding factor cascade and that other mechanisms might cause the observed nuclear translocation of beta-catenin.

20/3,AB/10 (Item 10 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

09775323 GENUINE ARTICLE#: 112EU NUMBER OF REFERENCES: 21
TITLE: beta-catenin *mutation"** in carcinoma of the uterine endometrium
AUTHOR(S): Fukuchi T; Sakamoto M; Tsuda H; Maruyama K; Nozawa S; Hirohashi S (REPRINT)
CORPORATE SOURCE: NATL CANC CTR,RES INST, DIV PATHOL, CHUO KU, 5-1-1 TSUKIJI/TOKYO 104//JAPAN/ (REPRINT); NATL CANC CTR,RES INST, DIV

PATHOL, CHUO KU/TOKYO 104//JAPAN/; KEIO UNIV,SCH MED, DEPT OBSTET & GYNECOL/TOKYO 160//JAPAN/
PUBLICATION TYPE: JOURNAL
PUBLICATION: CANCER RESEARCH, 1998, V58, N16 (AUG 15), P3526-3528
PUBLISHER: AMER ASSOC CANCER RESEARCH, PO BOX 11806, BIRMINGHAM, AL 35202
ISSN: 0008-5472
LANGUAGE: English DOCUMENT TYPE: ARTICLE
ABSTRACT: beta-Catenin forms complexes with *Tcf"** and Lef-*1"** and functions as a transcriptional activator downstream of the Wnt signaling pathway. Activation of the pathway by stabilization of beta-catenin has been shown to be important in the development of colorectal carcinoma, which is mainly caused by inactivating *mutations"** of the adenomatous polyposis coli tumor suppressor *gene"** or by activating *mutations"** in *exon"** 3 of the beta-catenin *gene"**. Here, we analyzed *mutations"** in *exon"** 3 of the beta-catenin *gene"** in endometrial carcinoma cases in which loss of heterozygosity at the adenomatous polyposis coli turner suppressor *gene"** locus has been rarely reported. We found that 10 of 76 cases had beta-catenin *gene"** *mutations"**. All *mutations"** identified were single-best! missense *mutations"** on serine/threonine residues (codons 33, 37, 41, and 45), altering the glycogen synthase kinase-3 beta phosphorylation consensus motif, which participates in the degradation of beta-catenin, To determine whether these beta-catenin *mutations"** actually led to stabilization of this protein, expression of beta-catenin was analyzed immunohistochemically, and 9 of 10 cases with the beta-catenin *mutation"** and 20 of 66 cases without it showed accumulation of beta-catenin in the cytoplasm and/or nucleus, In total, 38% of cases showed accumulation of beta-catenin. These data indicate that stabilization of beta-catenin due to *mutations"** in *exon"** 3 of the beta-catenin *gene"** and other mechanisms may have an important role in development of endometrial carcinomas.

20/3,AB/11 (Item 11 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

09488818 GENUINE ARTICLE#: ZP144 NUMBER OF REFERENCES: 32
TITLE: Genomic organization of the segment polarity *gene"** pan in Drosophila melanogaster
AUTHOR(S): Dooijes D; vanBeest M; vandeWetering M; Boulanger G; Jones T; Clevers H; Mortin MA (REPRINT)
CORPORATE SOURCE: NCI,BIOCHEM LAB, NIH, 37 CONVENT DR, BLDG 37, RM 4D-25/BETHESDA//MD/20892 (REPRINT); NCI,BIOCHEM LAB, NIH/BETHESDA//MD/20892; UNIV UTRECHT HOSP,DEPT IMMUNOL/NL-3508 GA UTRECHT//NETHERLANDS/
PUBLICATION TYPE: JOURNAL
PUBLICATION: MOLECULAR & GENERAL GENETICS, 1998, V258, N1-2 (APR), P45-52
PUBLISHER: SPRINGER VERLAG, 175 FIFTH AVE, NEW YORK, NY 10010
ISSN: 0026-8925
LANGUAGE: English DOCUMENT TYPE: ARTICLE
ABSTRACT: We previously described the molecular cloning of a mammalian T cell factor *1"** (*TCF"**-*1"**)-like protein from Drosophila melanogaster, encoded by the pangolin (pan) locus, and demonstrated that it consists of a DNA binding domain similar to that of other high mobility group proteins and a protein-protein interaction domain that binds beta-catenin (Armadillo in Drosophila) but that it lacks a transcriptional activation domain. Here we show that the pan locus

spans approximately 50 kb and the mRNA results from the splicing of 13 *exons"**. We note remarkable conservation of the *exon"**/intron boundaries between the human and D. melanogaster *genes"**, suggesting that they share a common ancestor. Chromosomal in situ hybridization locates pan to the base of chromosome 4, near the cubitus interruptus locus. Restriction map and sequence analyses confirm their close proximity. The small fourth chromosome undergoes little or no recombination and was previously reported to lack DNA *polymorphisms"** ; however, we note two DNA *polymorphisms"** occurring in three combinations within the pan locus, demonstrating the presence of synonymous substitutions and the past occurrence of recombination. We present evidence suggesting that the protein encoded by pan is more similar to mammalian *TCF"**-*1"** and Caenorhabditis elegans POP-1 than to mammalian LEF-1.

20/3,AB/12 (Item 12 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

08946612 GENUINE ARTICLE#: YD481 NUMBER OF REFERENCES: 38
TITLE: Analysis of the glucokinase *gene"** in Mexican families displaying early-onset non-insulin-dependent diabetes mellitus including MODY families
AUTHOR(S): delBosquePlata L; GarciaGarcia E; RamirezJimenez S; CabelloVillegas J; Riba L; GomezLeon A; VegaHernandez G; AltamiranoBustamante N; CalzadaLeon R; RoblesValdes C; MendozaMorfin F; CurielPerez O; TusieLuna MT (REPRINT)
CORPORATE SOURCE: INST NACL PEDIAT,APTO POSTAL 101-48/MEXICO CITY 04530/DF/MEXICO/ (REPRINT); INST NACL PEDIAT,/MEXICO CITY 04530/DF/MEXICO/; UNIV NACL AUTONOMA MEXICO,INST INVEST BIOMED, UNIDAD GENET/MEXICO CITY 04510/DF/MEXICO/; INST NACL NUTR SALVADOR ZUBIRAN,DIABET CLIN/MEXICO CITY 14000/DF/MEXICO/; UNIV NACL AUTONOMA MEXICO,FAC MED/MEXICO CITY 04510/DF/MEXICO/; UNIV NACL AUTONOMA MEXICO,DIRECC GEN COMPUTO ACAD/MEXICO CITY 04510/DF/MEXICO/; INST NACL PEDIAT,SERV ENDOCRINOL/MEXICO CITY/DF/MEXICO/; INST NACL PEDIAT,SERV ESPECIALIDADES MED/MEXICO CITY/DF/MEXICO/; INST MEXICANO SEGURO SOCIAL,GEN HOSP, CTR MED NACL, DEPT ENDOCRINOL/MEXICO CITY/DF/MEXICO/
PUBLICATION TYPE: JOURNAL
PUBLICATION: AMERICAN JOURNAL OF MEDICAL GENETICS, 1997, V72, N4 (NOV 12) , P387-393
PUBLISHER: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012
ISSN: 0148-7299
LANGUAGE: English DOCUMENT TYPE: ARTICLE
ABSTRACT: Non-insulin-dependent diabetes mellitus (NIDDM) is the most common form of diabetes, affecting 5% of the general population, Genetic factors play an important role in the development of the disease, While in other populations NIDDM is usually diagnosed after the fifth decade of life, in Mexico a large proportion of patients develop the disease at an early age (between the third and the fourth decade), In Caucasian population, *mutations"** in the glucokinase *gene"**, the *TCF1"**, and TCF14 *genes"**, have been identified in a subgroup of early-onset NIDDM patients denominated MODY (maturity-onset diabetes of the young), which show an autosomal dominant pattern of inheritance, As a first step in the molecular characterization of Mexican families displaying early-onset NIDDM we searched for *mutations"** in the glucokinase *gene"** through SSCP analysis and/or

direct sequencing in 26 individuals from 22 independent families, where at least four can be classified as MODY. No *mutations"** were detected in the *exons"** or the intron-*exon"** boundaries of the *gene"** in any of the screened individuals, The phenotype and clinical profile of some of the studied patients is compatible with that of patients carrying *mutations"** in the *TCF1"** or TCF14 *genes"**, while others may carry *mutations"** in different loci, Through computer simulation analysis we identified at least four informative families which will be used for further linkage studies. (C) 1997 Wiley-Liss, Inc.

20/3,AB/13 (Item 13 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

08935056 GENUINE ARTICLE#: YD172 NUMBER OF REFERENCES: 30
TITLE: TGF-beta 1 in colonic neoplasia: a genetic molecular and immunohistochemical study
AUTHOR(S): Cardillo MR (REPRINT); Yap E
CORPORATE SOURCE: UNIV ROMA LA SAPIENZA,SECT HISTOPATHOL, DEPT EXPT MED & PATHOL, VIALE REGINA ELENA 325/I-00161 ROME//ITALY/ (REPRINT)
PUBLICATION TYPE: JOURNAL
PUBLICATION: JOURNAL OF EXPERIMENTAL & CLINICAL CANCER RESEARCH, 1997, V16 , N3 (SEP), P281-288
PUBLISHER: APSIT ASSOC PROM STUD IMMUNOL TUMOR, VIALE REGINA ELENA 291, 00161 ROME, ITALY
ISSN: 0392-9078
LANGUAGE: English DOCUMENT TYPE: ARTICLE
ABSTRACT: Transforming growth factor-beta proteins are key regulators of cellular growth and differentiation. To understand the role of TGF-P in colonic tumour progression, 47 paraffin embedded samples from colonic tumours (13 adenomas, and 34 adenocarcinomas) were studied. *Gene"** *mutations"** in the region coding for the active protein were studied by PCR SSCP analysis of *exons"** 5, 6, and 7. TGF-beta 1 mRNA expression and localization were studied by NISH using cDNA probes generated by RT-PCR, Protein distribution was investigated by immunohistochemistry using antibodies against both intracellular and extracellular forms. Three *mutations"** were found: one in *exon"** 5 (Dukes C) and two in *exon"** 7 (Dukes C and A). TGF-beta 1 mRNA was observed in 9 (69%) of 13 adenomas and in 30 (88%) of 34 adenocarcinomas. Levels of TGF-beta 1 mRNA and proteins were higher in colorectal carcinomas than in colorectal adenomas. Tubular adenomas associated with dysplasia showed greater expression of TGF-beta 1 than adenomas without dysplasia and than non-neoplastic colonic mucosa. In dysplasia and cancer, epithelial neoplastic cells and stromal cells were positive for TGF-beta 1. In normal tissue endothelial cells and granulocytes sporadically showed immunoreactivity for TGF-beta 1, whereas epithelial cells were all negative. The three *mutations"** in TGF-beta 1 *exons"** 5, 6 and 7 found in colorectal adenocarcinomas suggest that TGF-beta 1 *mutation"** may play a role in colorectal carcinogenesis and that the presence of the *mutant"** form contributes to the transformed state. Our two other findings, the loss of staining gradient in normal colonic mucosa and the higher levels of TGF-beta 1 mRNA and proteins in colorectal carcinomas than in colorectal adenomas, indicate that the de-regulation of *TCF"**-beta *1"** expression may occur as an early event in colorectal carcinogenesis. Although TGF-beta 1 expression is generally a reflection of cellular differentiation, tumour grade is not associated with TGF-beta 1 expression. Beside being

present in the epithelial cells of the colonic tumours, TGF-beta 1 mRNA also occurred in the stroma: its localization in the macrophages adjacent to tumour strongly suggests that TGF-beta 1 could be secreted by macrophages. This hypothesis should lead to new therapeutic strategies for colorectal carcinoma.

20/3,AB/14 (Item 14 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

08351257 GENUINE ARTICLE#: WT546 NUMBER OF REFERENCES: 23
TITLE: Identification of nine novel mutations in the hepatocyte nuclear factor 1 alpha gene associated with maturity-onset diabetes of the young (MODY3)
AUTHOR(S): Vaxillaire M; Rouard M; Yamagata K; Oda N; Kaisaki PJ; Boriraj VV; Chevre JC; Boccio V; Cox RD; Lathrop GM; Dussoix P; Philippe J; Timsit J; Charpentier G; Velho G; Bell GI; Froguel P (REPRINT)
CORPORATE SOURCE: INST PASTEUR,CNRS EP10, 1 RUE PROF CALMETTE, BP 245/F-59019 LILLE//FRANCE/ (REPRINT); INST PASTEUR,CNRS EP10/F-59019 LILLE//FRANCE/; CHU LILLE,/F-59019 LILLE//FRANCE/; WELLCOME TRUST CTR HUMAN GENET,/OXFORD OX3 7BN//ENGLAND/; UNIV CHICAGO,DEPT BIOCHEM & MOL BIOL/CHICAGO//IL/60637; UNIV CHICAGO,HOWARD HUGHES MED INST/CHICAGO//IL/60637; UNIV CHICAGO,DEPT MED/CHICAGO//IL/60637; UNIV GENEVA,HOP CANTONAL, UNITE DIABETOL CLIN/CH-1211 GENEVA 14//SWITZERLAND/; HOP NECKER ENFANTS MALAD,SERV IMMUNOL CLIN/F-75015 PARIS//FRANCE/; CTR HOSP GILLES DE CORBEIL,SERV ENDOCRINOL/F-91108 CORBEIL ESSONNES//FRANCE/; HOP ST LOUIS,INSERM U358/F-75010 PARIS//FRANCE/
PUBLICATION TYPE: JOURNAL
PUBLICATION: HUMAN MOLECULAR GENETICS, 1997, V6, N4 (APR), P583-586
PUBLISHER: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD, ENGLAND OX2 6DP
ISSN: 0964-6906
LANGUAGE: English DOCUMENT TYPE: ARTICLE
ABSTRACT: Maturity-onset diabetes of the young (MODY) is a genetically heterogeneous subtype of non-insulin-dependent diabetes mellitus (NIDDM) characterised by early onset, autosomal dominant inheritance and a primary defect in insulin secretion, Recent studies have shown that mutations in the two functionally related transcription factors, hepatocyte nuclear factor 4 alpha (HNF-4 alpha) and hepatocyte nuclear factor 1 alpha (HNF-1 alpha) are associated with the MODY1 and MODY3 forms of diabetes respectively, whereas mutations in the enzyme glucokinase are the cause of the MODY2 form, We have examined 10 unrelated Caucasian families in which MODY/NIDDM co-segregated with markers for MODY3 for *mutations"** in the HNF-1 alpha *gene"** (*TCF1"**), Ten different *mutations"** were observed in these families, all of which co-segregated with diabetes, There were no obvious relationships between the nature of the mutations observed (i,e, frameshift, nonsense, or missense) or their location in the gene with clinical features of diabetes (age at onset, severity) in these families, The mechanisms by which mutations in the HNF-1 alpha gene cause diabetes mellitus are unclear but might include abnormal pancreatic islet development during foetal life thereby limiting their later function, as well as impaired transcriptional regulation of genes that play a key role in normal pancreatic beta cell function.

20/3,AB/15 (Item 1 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07777325 Genuine Article#: 194VR Number of References: 0
Title: Identification of new *mutations"** in the *TCF"**-*1"** and *TCF"**-14 *genes"** in Mexican patients with early onset type 2 diabetes
Author(s): Ordonez MAL; AguilarSalinas CA; Reyes E; Arellano E; Guillen LE; Ramirez S; Dominguez A; GomezPerez FJ; Rull JA; Tusie T
Journal: DIABETES, 1999, V48, 1, P821-821
ISSN: 0012-1797 Publication date: 19990000
Publisher: AMER DIABETES ASSOC, 1660 DUKE ST, ALEXANDRIA, VA 22314
Language: English Document Type: MEETING ABSTRACT

20/3,AB/16 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

01998969 Genuine Article#: JT432 Number of References: 13
Title: THE *GENE"** CODING FOR *VARIANT"** HEPATIC NUCLEAR FACTOR-*I"** (*TCF"**-2), MAPS BETWEEN THE EDP-1 AND ERBA *GENES"** ON MOUSE CHROMOSOME-11
Author(s): KAROLYI IJ; GUENET JL; REYCAMPOS J; CAMPER SA
Corporate Source: INST PASTEUR,UNITE VIRUS ONCOGENES/F-75724 PARIS 15//FRANCE/; UNIV MICHIGAN,SCH MED,DEPT HUMAN GENET/ANN ARBOR//MI/48109 ; INST PASTEUR,UNITE GENET MAMMIFERES/F-75724 PARIS 15//FRANCE/
Journal: MAMMALIAN GENOME, 1992, V3, N3, P184-185
ISSN: 0938-8990
Language: ENGLISH Document Type: NOTE

20/3,AB/17 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11354326 21183105 PMID: 11289470
Role of common sequence *variants"** in insulin secretion in familial type 2 diabetic kindreds: the sulfonylurea receptor, glucokinase, and hepatocyte nuclear factor 1alpha *genes"**.
Elbein SC; Sun J; Scroggin E; Teng K; Hasstedt SJ
Department of Medicine, Central Arkansas Veterans Healthcare System and University of Arkansas for Medical Sciences, Little Rock, USA. elbeinstevenc@exchange.uams.edu
Diabetes care (United States) Mar 2001, 24 (3) p472-8, ISSN 0149-5992 Journal Code: EAG
Contract/Grant No.: DK39311, DK, NIDDK; M01-RR00064, RR, NCRR
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
OBJECTIVE: We have demonstrated high heritability of insulin secretion measured as acute insulin response to glucose times insulin sensitivity (disposition index). Furthermore, we showed that obese normoglycemic family members of a type 2 diabetic proband failed to compensate for the insulin resistance of obesity by increasing insulin secretion. In this study, we tested the primary hypotheses that previously described *variants"** in the pancreatic sulfonylurea receptor *gene"** (SUR1 or ABCC8), glucokinase (GCK) *gene"**, or hepatocyte nuclear factor 1alpha (*TCF1"** or HNF1alpha) *gene"** contribute to the inherited deficiencies of insulin secretion and beta-cell compensation to insulin resistance, as well as the secondary

hypotheses that these *variants"** altered insulin sensitivity. RESEARCH DESIGN AND METHODS: We typed 124 nondiabetic members of 26 familial type 2 diabetic kindreds who had undergone tolbutamide-modified intravenous glucose tolerance tests for two *variants"** of the ABCC8 (sulfonylurea) *gene"**, two *variants"** of the GCK *gene"**, and one common amino acid *variant"** in the *TCF1"** (HNF1alpha) *gene"**. All family members were classified as normal or having impaired glucose tolerance based on oral glucose tolerance testing. We used minimal model analysis to calculate the insulin sensitivity index (S1) and glucose effectiveness (SG), and acute insulin response to glucose was calculated as the mean insulin excursion above baseline during the first 10 min after the glucose bolus. Disposition index (DI), a measure of beta-cell compensation for insulin sensitivity, was calculated as insulin sensitivity times acute insulin response. Effects of *polymorphisms"** were determined using mixed effects models that incorporated family membership and by a likelihood analysis that accounted for family structure through polygenic inheritance. RESULTS: An intronic *variant"** of the ABCC8 *gene"** just upstream of *exon"** 16 was a significant determinant of both DI and an analogous index based on acute insulin response to tolbutamide. Surprisingly, heterozygous individuals showed the lowest indexes, whereas the DI in the two homozygous states did not differ significantly. Neither the *exon"** 18 *variant"** nor the *variants"** in the GCK and *TCF1"** *genes"** were significant in this model. However, combined genotypes of ABCC8 *exon"** 16 and 18 *variants"** again significantly predicted both indexes of glucose and tolbutamide-stimulated insulin secretion. Unexpectedly, a *variant"** in the 3' untranslated region of the GCK *gene"** interacted significantly with BMI to predict insulin sensitivity. CONCLUSIONS: The *exon"** 16 *variant"** of the ABCC8 *gene"** reduced beta-cell compensation to the decreased insulin sensitivity in the heterozygous state. This may explain the observation from several groups of an association of the ABCC8 *variants"** in diabetes and is consistent with other studies showing a role of ABCC8 *variants"** in pancreatic beta-cell function. However, our study focused on individuals from relatively few families. Ascertainment bias, family structure, and other interacting *genes"** might have influenced our unexpected result. Additional studies are needed to replicate our observed deficit in beta-cell compensation in individuals heterozygous for ABCC8 *variants"**. Likewise, the role of the GCK 3' *variant"** in the reduced insulin sensitivity of obesity will require further study.

20/3,AB/18 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10503706 20152818 PMID: 10690959
Molecular scanning analysis of hepatocyte nuclear factor 1alpha (*TCF1"**) *gene"** in typical familial type 2 diabetes in African Americans.
Elbein SC; Teng K; Eddings K; Hargrove D; Scroggin E
Division of Endocrinology, Central Arkansas Veterans Health System and University of Arkansas for Medical Sciences, Little Rock, USA.
Metabolism: clinical and experimental (UNITED STATES) Feb 2000, 49 (2) p280-4, ISSN 0026-0495 Journal Code: MUM
Contract/Grant No.: DK39301, DK, NIDDK
Languages: ENGLISH
Document type: Clinical Trial; Journal Article
Record type: Completed
Type 2 diabetes mellitus (T2DM) is strongly inherited, but the major *genes"** for this disease have been elusive. In contrast, early-onset,

autosomal-dominant diabetes results from at least 5 loci, of which hepatocyte nuclear factor 1a (HNF1alpha or *TCF1"**) is the most common cause. *Mutations"** in HNF1alpha also cause later-onset diabetes in some Caucasian populations, but the role of these *mutations"** has not been tested in African American populations. We used a variety of screening methods, including both single-strand conformation *polymorphism"** (SSCP) analysis and dideoxy fingerprint analysis, to search for *mutations"** in 51 African American subjects with onset of diabetes before age 50 years. Potential *mutations"** were confirmed by direct sequencing. We identified 21 different *variants"** , of which 11 were unique to African Americans. Four *mutations"** either altered the amino acid sequence (Gly52Ala and Gly574Ser) or were close to a splice site (intron 1 and intron 10). A 5-nucleotide insertion in intron 1 was present in both diabetic members of a small family, but Gly52Ala, Gly574Ser, and the intron 10 *mutation"** did not segregate with diabetes. Gly574Ser was present in 2 large families and 5% of controls, all of which appeared to share the same common HNF1alpha haplotype. Surprisingly, radioactive SSCP analysis under 2 room-temperature conditions performed as well as methods using fluorescent labeling that were expected to be more sensitive. We conclude that in African American individuals under age 50, variation in the HNF1a *gene"** is common but unlikely to be a significant cause of T2DM.

20/3,AB/19 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10481337 20115343 PMID: 10649499
Big Dye terminator cycle sequencing chemistry: accuracy of the dilution process and application for screening *mutations"** in the *TCF1"** and GCK *genes"**.
Boutin P; Wahl C; Samson C; Vasseur F; Laget F; Froguel P
Human mutation (UNITED STATES) 2000, 15 (2) p201-3, ISSN 1059-7794 Journal Code: BRD
Languages: ENGLISH
Document type: Letter
Record type: Completed

20/3,AB/20 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09946030 98425814 PMID: 9754819
*Mutation"** screening in 18 Caucasian families suggest the existence of other MODY *genes"**.
Chevre JC; Hani EH; Boutin P; Vaxillaire M; Blanche H; Vionnet N; Pardini VC; Timsit J; Larger E; Charpentier G; Beckers D; Maes M; Bellanne-Chantelot C; Velho G; Froguel P
Institut de Biologie de Lille, CNRS EP-10, France.
Diabetologia (GERMANY) Sep 1998, 41 (9) p1017-23, ISSN 0012-186X Journal Code: E93
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Maturity-onset diabetes of the young (MODY) is a heterogeneous subtype of non-insulin-dependent diabetes mellitus characterised by early onset, autosomal dominant inheritance and a primary defect in insulin secretion. To date five MODY *genes"** have been identified: hepatocyte nuclear factor-4 alpha (HNF-4alpha/MODY1/TCF14) on chromosome 20q, glucokinase

(GCK/MODY2) on chromosome 7p, hepatocyte nuclear factor-1 alpha (HNF-1alpha/MODY3/*TCF1"**) on chromosome 12q, insulin promoter factor-1 (IPF1/MODY4) on chromosome 13q and hepatocyte nuclear factor-1 beta (HNF-1beta/MODY5/TCF2) on chromosome 17cen-q. We have screened the HNF-4alpha, HNF-1alpha and HNF-1beta *genes"** in members of 18 MODY kindreds who tested negative for glucokinase *mutations"**. Five missense (G31D, R159W, A161T, R200W, R271W), one substitution at the splice donor site of intron 5 (IVS5nt + 2T-->A) and one deletion *mutation"** (P379fsdelT) were found in the HNF-1alpha *gene"**, but no MODY-associated *mutations"** were found in the HNF-4alpha and HNF-1beta *genes"**. Of 67 French MODY families that we have now studied, 42 (63%) have *mutations"** in the glucokinase *gene"**, 14 (21%) have *mutations"** in the HNF-1alpha *gene"**, and 11 (16%) have no *mutations"** in the HNF-4alpha, IPF1 and HNF-1beta *genes"**. Eleven families do not have *mutations"** in the five known MODY *genes"** suggesting that there is at least one additional locus that can cause MODY.

20/3,AB/21 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09479877 98041566 PMID: 9375718

Analysis of the glucokinase *gene"** in Mexican families displaying early-onset non-insulin-dependent diabetes mellitus including MODY families.

del Bosque-Plata L; Garcia-Garcia E; Ramirez-Jimenez S; Cabello-Villegas J; Riba L; Gomez-Leon A; Vega-Hernandez G; Altamirano-Bustamante N; Calzada-Leon R; Robles-Valdes C; Mendoza-Morfin F; Curiel-Perez O; Tusie-Luna MT

Unidad de Genetica de la Nutricion del Instituto de Investigaciones Biomedicas, Universidad Nacional Autonoma de Mexico y del Instituto Nacional de Pediatria, Mexico City, Mexico.

American journal of medical genetics (UNITED STATES) Nov 12 1997, 72 (4) p387-93, ISSN 0148-7299 Journal Code: 3L4

Languages: ENGLISH

Document type: Journal Article

Record type: Completed

Non-insulin-dependent diabetes mellitus (NIDDM) is the most common form of diabetes, affecting 5% of the general population. Genetic factors play an important role in the development of the disease. While in other populations NIDDM is usually diagnosed after the fifth decade of life, in Mexico a large proportion of patients develop the disease at an early age (between the third and the fourth decade). In Caucasian population, *mutations"** in the glucokinase *gene"**, the *TCF1"**, and TCF14 *genes"**, have been identified in a subgroup of early-onset NIDDM patients denominated MODY (maturity-onset diabetes of the young), which show an autosomal dominant pattern of inheritance. As a first step in the molecular characterization of Mexican families displaying early-onset NIDDM we searched for *mutations"** in the glucokinase *gene"** through SSCP analysis and/or direct sequencing in 26 individuals from 22 independent families, where at least four can be classified as MODY. No *mutations"** were detected in the *exons"** or the intron-*exon"** boundaries of the *gene"** in any of the screened individuals. The phenotype and clinical profile of some of the studied patients is compatible with that of patients carrying *mutations"** in the *TCF1"** or TCF14 *genes"**, while others may carry *mutations"** in different loci. Through computer simulation analysis we identified at least four informative families which will be used for further linkage studies.

20/3,AB/22 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08009375 93360980 PMID: 8102789
A novel POU domain protein which binds to the T-cell receptor beta enhancer.
Messier H; Brickner H; Gaikwad J; Fotedar A
Division of Molecular Biology, La Jolla Institute for Allergy and Immunology, California 92037.
Molecular and cellular biology (UNITED STATES) Sep 1993, 13 (9) p5450-60, ISSN 0270-7306 Journal Code: NGY
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
POU domain proteins have been implicated in the regulation of a number of lineage-specific *genes"** . Among the first POU domain proteins described were the immunoglobulin octamer-binding proteins Oct-1 and Oct-2. It was therefore of special interest when we identified a novel lymphoid POU domain protein in Southwestern (DNA-protein) screens of T-cell lambda gt11 libraries. This novel POU protein, *TCF"** beta *1"** , binds in a sequence-specific manner to a critical motif in the T-cell receptor (TCR) beta enhancer. Sequence analysis revealed that *TCF"** beta *1"** represents a new class of POU domain proteins which are distantly related to other POU proteins. *TCF"** beta *1"** is encoded by multiple *exons"** whose organization is distinct from that of other POU domain proteins. The expression of *TCF"** beta *1"** in a tissue-restricted manner and its ability to bind to multiple motifs in the TCR beta enhancer support a role in regulating TCR beta *gene"** expression. The expression of *TCF"** beta *1"** in both B and T cells and the ability of recombinant *TCF"** beta *1"** to bind octamer and octamer-related motifs suggest that *TCF"** beta *1"** has additional roles in lymphoid cell function. The ability of *TCF"** beta *1"** to transactivate in a sequence-specific manner is consistent with a role for regulating lymphoid *gene"** expression.

20/3,AB/23 (Item 1 from file: 76)
DIALOG(R)File 76:Life Sciences Collection

02608480 5141956
beta -catenin-sensitive isoforms of lymphoid enhancer factor-1 are selectively expressed in colon cancer
Hovanes, K.; Li, T.W.H.; Munguia, J.E.; Troung, Trung; Milovanovic, T.; Marsh, J.L.; Holcombe, R.F.; Waterman, M.L.
Microbiology and Molecular Genetics Department, University of California, Irvine, Irvine, California, USA
Nature Genetics vol. 28, no. 1, pp. 53-57 (2001)
ISSN: 1061-4036
DOCUMENT TYPE: Journal article LANGUAGE: ENGLISH
SUBFILE: Genetics Abstracts

Constitutive activation of the Wnt signaling pathway is a root cause of many colon cancers. Activation of this pathway is caused by genetic mutations that stabilize the beta -catenin protein, allowing it to accumulate in the nucleus and form complexes with any member of the lymphoid enhancer factor (LEF1) and T-cell factor (TCF1, TCF3, TCF4) family

of transcription factors (referred to collectively as LEF/TCFs) to activate transcription of target genes. Target genes such as MYC, CCND1, MMP7 and TCF7 (refs. 59) are normally expressed in colon tissue, so it has been proposed that abnormal expression levels or patterns imposed by beta -catenin/TCF complexes have a role in tumor progression. We report here that LEF1 is a new type of target gene ectopically activated in colon cancer. The pattern of this ectopic expression is unusual because it derives from selective activation of a promoter for a full-length LEF1 isoform that binds beta -catenin, but not a second, intronic promoter that drives expression of a dominant-negative isoform. beta -catenin/TCF complexes can activate the promoter for full-length LEF1, indicating that in cancer high levels of these complexes misregulate transcription to favor a positive feedback loop for Wnt signaling by inducing selective expression of full-length, beta -catenin-sensitive forms of LEF/TCFs.

20/3,AB/24 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

✓ INV

136113776 CA: 136(8)113776c PATENT
TCF-1 gene polymorphic allele A (with mutation C883.fwdarw.A) associated with Th1 and Th2 diseases and therapeutic and diagnostic methods thereof
INVENTOR(AUTHOR): Begovich, Ann Bethea; Erlich, Henry Anthony; Gruppe, Andrew; Noble, Janelle Annette; Peltz, Gary Allen; Reynolds, Rebecca Lynne; Walker, Karen Myra; Zangenberg, Gabriele
LOCATION: Germany,
ASSIGNEE: Roche Diagnostics G.m.b.H.; F. Hoffmann-La Roche A.-G.
PATENT: European Pat. Appl. ; EP 1174522 A2 DATE: 20020123
APPLICATION: EP 2001116692 (20010717) *US PV219812 (20000721)
PAGES: 38 pp. CODEN: EPXXDW LANGUAGE: English CLASS: C12Q-001/68A
DESIGNATED COUNTRIES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IT; LI; LU; NL; SE; MC; PT; IE; SI; LT; LV; FI; RO

20/3,AB/25 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

134145522 CA: 134(11)145522s JOURNAL
Mutations of adenomatous polyposis coli and .beta.-catenin genes during progression of lung tumors induced by N-nitrosobis(2-hydroxypropyl)amine in rats
AUTHOR(S): Tsujiuchi, Toshifumi; Tsutsumi, Masahiro; Sasaki, Yasutaka; Murata, Nao; Konishi, Yoichi
LOCATION: Department of Oncological Pathology, Cancer Center, Nara Medical University, Nara, Japan, 634-8521
JOURNAL: Cancer Res. DATE: 2000 VOLUME: 60 NUMBER: 23 PAGES: 6611-6616 CODEN: CNREA8 ISSN: 0008-5472 LANGUAGE: English PUBLISHER: American Association for Cancer Research

20/3,AB/26 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

131320891 CA: 131(24)320891y JOURNAL
Synergy between tumor suppressor APC and the .beta.-catenin-Tcf4 target

Tcf1
AUTHOR(S): Roose, Jeroen; Huls, Gerwin; van Beest, Moniek; Moerer, Petra; van der Horn, Karin; Goldschmeding, Roet; Logtenberg, Ton; Clevers, Hans
LOCATION: Department of Immunology and Center for Biomedical Genetics, University Medical Center Utrecht, 3508 GA, Utrecht, Neth.
JOURNAL: Science (Washington, D. C.) DATE: 1999 VOLUME: 285 NUMBER: 5435 PAGES: 1923-1926 CODEN: SCIEAS ISSN: 0036-8075 LANGUAGE: English
PUBLISHER: American Association for the Advancement of Science

20/3,AB/27 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

130310058 CA: 130(23)310058x JOURNAL
Identification of mutations in the hepatocyte nuclear factor-1.alpha. gene in Japanese subjects with early-onset NIDDM and functional analysis of the mutant proteins
AUTHOR(S): Yamada, Shirou; Tomura, Hideaki; Nishigori, Hidekazu; Sho, Kimie; Mabe, Hiroyo; Iwatani, Noritaka; Takumi, Toru; Kito, Yoshihiko; Moriya, Noaki; Muroya, Koji; Ogata, Tsutomu; Onigata, Kazumichi; Morikawa, Akihiro; Inoue, Ituro; Takeda, Jun
LOCATION: Laboratory of Molecular Genetics, Department of Cell Biology, Institute for Molecular and Cellular Regulation, Gunma, Japan, 371-8512
JOURNAL: Diabetes DATE: 1999 VOLUME: 48 NUMBER: 3 PAGES: 645-648
CODEN: DIAEAZ ISSN: 0012-1797 LANGUAGE: English PUBLISHER: American Diabetes Association

20/3,AB/28 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

127276442 CA: 127(20)276442u JOURNAL
Mutations in the hepatocyte nuclear factor-1.alpha. gene (MODY3) are not a major cause of late-onset NIDDM in Japanese subjects
AUTHOR(S): Yamada, Shirou; Nishigori, Hidekazu; Onda, Hideaki; Takahashi, Ken-Ichiro; Kitano, Norikazu; Morikawa, Akihiro; Takeuchi, Toshiyuki; Takeda, Jun
LOCATION: Departments of Molecular Genetics, Institute for Molecular and Cellular Regulation, Gunma University, Maebashi, Japan, 371
JOURNAL: Diabetes DATE: 1997 VOLUME: 46 NUMBER: 9 PAGES: 1512-1513
CODEN: DIAEAZ ISSN: 0012-1797 LANGUAGE: English PUBLISHER: American Diabetes Association, Inc.

20/3,AB/29 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

127276441 CA: 127(20)276441t JOURNAL
Mutations in the hepatocyte nuclear factor-1.alpha./MODY3 gene in Japanese subjects with early- and late-onset NIDDM
AUTHOR(S): Iwasaki, Naoko; Oda, Naohisa; Ogata, Makiko; Hara, Manami; Hinokio, Yoshinori; Oda, Yukie; Yamagata, Kazuya; Kanematsu, Sachiko; Ohgawara, Hisako; Omori, Yasue; Bell, Graeme I.
LOCATION: Diabetes Center, Tokyo Women's Medical College, Tokyo, Japan, 162

JOURNAL: Diabetes DATE: 1997 VOLUME: 46 NUMBER: 9 PAGES: 1504-1508
CODEN: DIAEAZ ISSN: 0012-1797 LANGUAGE: English PUBLISHER: American Diabetes Association, Inc.

20/3,AB/30 (Item 7 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

127079655 CA: 127(6)79655q JOURNAL
Mutations in the hepatocyte nuclear factor-1.alpha. gene in MODY and early-onset NIDDM: evidence for a mutational hotspot in exon. (Erratum to document cited in CA126:262647)
AUTHOR(S): Kaisaki, Pamela J.; Menzel, Stephan; Lindner, Tom; Oda, Naohisa; Rjasanowski, Ilona; Sahm, Juergen; Meincke, Gustav; Schulze, Jan; Schmechel, Harald; Petzold, Cornelia; Ledermann, Hellmuth M.; Sachse, Guenther; Boriraj, V. Vicky; Menzel, Ruth; Kerner, Wolfgang; Turner, Robert C.; Yamagata, Kazuya; Bell, Graeme I.
LOCATION: Howard Hughes Medical Inst., Univ. Chicago, Chicago, IL, 60637, USA
JOURNAL: Diabetes DATE: 1997 VOLUME: 46 NUMBER: 7 PAGES: 1239
CODEN: DIAEAZ ISSN: 0012-1797 LANGUAGE: English PUBLISHER: American Diabetes Association, Inc.

20/3,AB/31 (Item 8 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

127064024 CA: 127(5)64024j JOURNAL
Novel mutations and a mutational hotspot in the MODY3 gene
AUTHOR(S): Glucksmann, M. Alexandra; Lehto, Markku; Tayber, Olga; Scotti, Susan; Berkemeier, Lucy; Pulido, Jacqueline C.; Wu, Ye; Nir, Waan-Jeng; Fang, Lei; Markel, Paul; Munnelly, Kevin D.; Goranson, Jill; Orho, Marju; Young, Brian M.; Whitacre, Jennifer L.; McMenimen, Cheryl; Wantman, Michael ; Tuomi, Tiinamaija; Warram, James; Forsblom, Carol M.; Carlsson, Martin; Rosenzweig, James; Kennedy, Giulia; Duyk, Geoffrey M.; Krolewski, Andrzej S.; Groop, Leif C.; Thomas, Jeffrey D.
LOCATION: Millennium Pharmaceuticals, Inc., Cambridge, MA, USA
JOURNAL: Diabetes DATE: 1997 VOLUME: 46 NUMBER: 6 PAGES: 1081-1086
CODEN: DIAEAZ ISSN: 0012-1797 LANGUAGE: English PUBLISHER: American Diabetes Association, Inc.

20/3,AB/32 (Item 9 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

126262647 CA: 126(20)262647g JOURNAL
Mutations in the hepatocyte nuclear factor-1.alpha. gene in MODY and early-onset NIDDM. Evidence for a mutational hotspot in exon 4
AUTHOR(S): Kaisaki, Pamela J.; Menzel, Stephan; Lindner, Tom; Oda, Naohisa; Rjasanowski, Ilona; Sahm, Juergen; Meincke, Gustav; Schulze, Jan; Schmechel, Harald; Petzold, Cornelia; Ledermann, Hellmuth M.; Sachse, Guenther; Boriraj, V. Vicky; Menzel, Ruth; Kerner, Wolfgang; Turner, Robert C.; Yamagata, Kazuya; Bell, Graeme I.
LOCATION: Howard Hughes Medical Inst., Univ. Chicago, Chicago, IL, 60637, USA

JOURNAL: Diabetes DATE: 1997 VOLUME: 46 NUMBER: 3 PAGES: 528-535
CODEN: DIAEAZ ISSN: 0012-1797 LANGUAGE: English PUBLISHER: American Diabetes Association, Inc.

20/3,AB/33 (Item 10 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

126017356 CA: 126(2)17356p JOURNAL
Mutations in the hepatocyte nuclear factor-1.alpha. gene in maturity-onset diabetes of the young (MODY3)
AUTHOR(S): Yamagata, Kazuya; Oda, Naohisa; Kaisaki, Pamela J.; Menzel, Stephan; Furuta, Hiroto; Vaxillaire, Martine; Southam, Lorraine; Cox, Roger D.; Lathrop, G. Mark; Boriraj, V. Vicky; Chen, Xiangna; Cox, Nancy J.; Oda, Yukie; Yano, Hideki; Le Beau, Michelle M.; Yamada, Shirou; Nishigori, Hidekazu; Takeda, Jun; Fajans, Stefan S.; Hattersley, Andrew T.; Iwasaki, Naoko; Hansen, Torben; Pedersen, Oluf; Polonsky, Kenneth S.; Turner, Robert C.; Velho, Gilberto; Chevre, Jean-Claude; Froguel, Philippe; Bell, Graeme I.
LOCATION: Howard Hughes Medical Inst., Univ. Chicago, Chicago, IL, 60637, USA
JOURNAL: Nature (London) DATE: 1996 VOLUME: 384 NUMBER: 6608 PAGES: 455-458 CODEN: NATUAS ISSN: 0028-0836 LANGUAGE: English PUBLISHER: Macmillan Magazines

20/3,AB/34 (Item 11 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

121028219 CA: 121(3)28219u JOURNAL
Naturally occurring genotype of the human immunodeficiency virus type 1 long terminal repeat display a wide range of basal and Tat-induced transcriptional activities
AUTHOR(S): Michael, Nelson L.; D'Arcy, Lisa; Ehrenberg, Philip K.; Redfield, Robert R.
LOCATION: Henry M. Jackson Foundat., Walter Reed Army Inst. Res., Rockville, MD, USA
JOURNAL: J. Virol. DATE: 1994 VOLUME: 68 NUMBER: 5 PAGES: 3163-74
CODEN: JOVIAM ISSN: 0022-538X LANGUAGE: English

20/3,AB/35 (Item 1 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

02040771 SUPPLIER NUMBER: 80392790 (USE FORMAT 7 OR 9 FOR FULL TEXT)
No evidence for linkage or for diabetes-associated *mutations"** in the activin type 2B receptor *gene"** (ACVR2B) in French patients with mature-onset diabetes of the young or type 2 diabetes. (Brief Genetics Report).(Statistical Data Included)
Dupont, Sophie; Hani, El Habib; Cras-Meneur, Corentin; De Matos, Frederique ; Lobbens, Stephane; Lecoeur, Cecile; Vaxillaire, Martine; Scharfmann, Raphael; Froguel, Philippe
Diabetes, 50, 5, 1219(3)
May,
2001

DOCUMENT TYPE: Statistical Data Included PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0012-1797 LANGUAGE: English
RECORD TYPE: Fulltext TARGET AUDIENCE: Professional
WORD COUNT: 2745 LINE COUNT: 00234

20/3,AB/36 (Item 2 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01662458 SUPPLIER NUMBER: 18965500 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Surprising pair of diabetes genes debuts. (genes that cause some cases of diabetes identified)
Travis, John
Science News, v150, n23, p359(1)
Dec 7,
1996
PUBLICATION FORMAT: Magazine/Journal ISSN: 0036-8423 LANGUAGE: English
RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Academic; Consumer
WORD COUNT: 706 LINE COUNT: 00059

ABSTRACT: Researchers have identified the genes responsible for maturity-onset diabetes of the young (MODY). Although MODY is a rare form of diabetes, the research may have implications for the prevention and treatment of all types of diabetes.

20/3,AB/37 (Item 3 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01508415 SUPPLIER NUMBER: 16955223
An HMG-box-containing T-cell factor required for thymocyte differentiation.
Verbeek, Sjef; Izon, David; Hofhuis, Frans; Robanus-Maandag, Els; Riele, Hein te; Wetering, Marc van de; Oosterwegel, Mariette; Wilson, Anne; MacDonald, H. Robson; Clevers, Hans
Nature, v374, n6517, p70(5)
March 2,
1995
PUBLICATION FORMAT: Magazine/Journal ISSN: 0028-0836 LANGUAGE: English
RECORD TYPE: Abstract TARGET AUDIENCE: Academic

ABSTRACT: A study of mice containing *mutations"** in T-cell factor-*1"** (*Tcf"**-*1"**), a thymocyte differentiation regulating *gene"**, indicates the absence of transition of the thymocytes from the CD8+ stage to the CD4+/CD8+ stage, which is a crucial step in cell differentiation, in the mutant mice. This implies the importance of Tcf-1, which encodes HMG-box proteins, for cell differentiation. The mutants exhibit low levels of immunocompetent T cells in the peripheral lymphoid organs.

20/3,AB/38 (Item 1 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS

01383032
TCF-1 nucleotide sequence variation
Tcf-1 Nukleotidsequenzvariation

Une variation de sequence nucleotidique de *TCF"**-*1"**
PATENT ASSIGNEE:
  Roche Diagnostics GmbH, (2638980), Sandhofer Strasse 116, 68305 Mannheim, DE\(Applicant designated states: , DE)
  F. HOFFMANN-LA ROCHE AG, (200573), Grenzacherstrasse 124, 4002 Basel, CH\(Applicant designated states: , BE; CH; DK; ES; FI; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE; AT; CY)
INVENTOR:
  Begovich, Ann Bethea, 7306 Rockway Avenue, El Cerrito, CA 94530, (US)
  Erlich, Henry Anthony, 3936 Rhoda Avenue, Oakland, CA 94602, (US)
  Gruppe, Andrew, 554 Jeter Street, Redwood City, California 94062, (US)
  Noble, Janelle Annette, 1430 Stannage Avenue, Berkeley, CA 94702, (US)
  Peltz, Gary Allen, 110 Danbury Lane, Redwood City, CA 94303, (US)
  Reynolds, Rebecca Lynne, 913 Independence Drive, Alameda, CA 94501-1045, (US)
  Walker, Karen Myra, 413 Haight Avenue, Alameda, CA 94501, (US)
  Zangenberg, Gabriele, Ringstrasse 16, 82319 Starnberg, (DE)
PATENT (CC, No, Kind, Date): EP 1174522 A2 020123 (Basic)
APPLICATION (CC, No, Date): EP 2001116692 010717;
PRIORITY (CC, No, Date): US 219812 000721
DESIGNATED STATES: AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE; TR
EXTENDED DESIGNATED STATES: AL; LT; LV; MK; RO; SI
INTERNATIONAL PATENT CLASS: C12Q-001/68

ABSTRACT EP 1174522 A2
  Methods and reagents for determining sequence variants present at the TCF-1 locus, which facilitates identifying individuals at risk for Th1 diseases, such as type 1 diabetes or multiple sclerosis, or Th2 diseases, such as allergic asthma or atopy.
ABSTRACT WORD COUNT: 39

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

Available Text	Language	Update	Word Count
CLAIMS A	(English)	200204	767
SPEC A	(English)	200204	15639
Total word count - document A			16406
Total word count - document B			0
Total word count - documents A + B			16406

20/3,AB/39 (Item 2 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS

01322386
Primers for synthesizing full length cDNA clones and their use
Primer zur Synthese von vollstandigen cDNA Klonen und ihre Verwendung
Amorces pour la synthese de cADN de pleine longueur et leur utilisation
PATENT ASSIGNEE:
  Helix Research Institute, (2656450), 1532-3 Yana, Kisarazu-shi, Chiba 292-0812, (JP), (Applicant designated States: all)
INVENTOR:
  Ota, Toshio, 1-2-7-105, Tsujido Shinmachi, Fujisawa-shi, Kanagawa 251-0042, (JP)
  Nishikawa, Tetsuo, 27-3-403, Hikawa-cho, Itabashi-ku, Tokyo 173-0013, (JP)

Isogai, Takao, 511-12, Ohmuro, Ami-machi, Inashiki-gun, Ibaraki 300-0303, (JP)
Hayashi, Koji, 1-9-446, Yushudai Nishi, Ichihara-shi, Chiba 299-0125, (JP)
Ishii, Shizuko, 4508-19-202, Yana, Kisarazu-shi, Chiba 292-0812, (JP)
Kawai, Yuri, 4508-19-201, Yana, Kisarazu-shi, Chiba 292-0812, (JP)
Wakamatsu, Ai, 1473-4-202, Takayanagi, Kisarazu-shi, Chiba 292-0014, (JP)
Sugiyama, Tomoyasu, 2-6-23-102, Kiyomidai, Kisarazu-shi, Chiba 292-0045, (JP)
Nagai, Keiichi, 3-44-14-9-204, Sakuragaoka, Higashiyamato-shi, Tokyo 207-0022, (JP)
Kojima, Shinichi, 2-7-10-202, Gion, Kisarazu-shi, Chiba 292-0052, (JP)
Otsuki, Tetsuji, 3-1-10-B102, Asahi, Kisarazu-shi, Chiba 292-0055, (JP)
Koga, Hisashi, 2-4-15, Asahi, Kisarazu-shi, Chiba 292-0055, (JP)
LEGAL REPRESENTATIVE:
VOSSIUS & PARTNER (100314), Siebertstrasse 4, 81675 Munchen, (DE)
PATENT (CC, No, Kind, Date): EP 1130094 A2 010905 (Basic)
EP 1130094 A3 011121
APPLICATION (CC, No, Date): EP 2000114089 000707;
PRIORITY (CC, No, Date): JP 99194486 990708; JP 2000118774 000111; JP 2000183765 000502
DESIGNATED STATES: AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE
EXTENDED DESIGNATED STATES: AL; LT; LV; MK; RO; SI
INTERNATIONAL PATENT CLASS: C12N-015/12; C12N-015/11; C12N-015/10; C12N-015/70; C12N-015/85; C12N-005/10; C12N-001/21; C07K-014/47; C07K-016/18; C12Q-001/68

ABSTRACT EP 1130094 A2
Primers for synthesizing full length cDNAs and their use are provided. 830 cDNA encoding a human protein has been isolated and nucleotide sequences of 5'-, and 3'-ends of the cDNA have been determined. Furthermore, primers for synthesizing the full length cDNA have been provided to clarify the function of the protein encoded by the cDNA. The full length cDNA of the present invention containing the translation start site provides information useful for analyzing the functions of the protein.
ABSTRACT WORD COUNT: 79
NOTE:
Figure number on first page: 1

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

Available Text	Language	Update	Word Count
CLAIMS A	(English)	200136	709
SPEC A	(English)	200136	97667
Total word count - document A			98376
Total word count - document B			0
Total word count - documents A + B			98376

20/3,AB/40 (Item 1 from file: 98)
DIALOG(R)File 98:General Sci Abs/Full-Text

04255630 H.W. WILSON RECORD NUMBER: BGSA00005630
Lentivirus replication and regulation.
AUGMENTED TITLE: review

Tang, Hengli
Kuhen, Kelli L; Wong-Staal, Flossie
Annual Review of Genetics v. 33 (1999) p. 133-70
SPECIAL FEATURES: bibl il ISSN: 0066-4197
LANGUAGE: English
COUNTRY OF PUBLICATION: United States
WORD COUNT: 17942

ABSTRACT: Lentiviruses are associated with chronic diseases of the hematological and neurological systems in animals and man. In particular, human immunodeficiency virus type 1 (HIV-1) is the etiological agent of the global AIDS epidemic. The genomes of lentiviruses are complex, encoding a number of regulatory and accessory proteins not found in other retroviruses. This complexity is reflected in their replication cycle, which reveals intricate regulatory pathways and unique mechanisms for viral persistence. In this review, we highlight some of these unique features for HIV-1, with particular focus on the transcriptional and posttranscriptional control of *gene"** expression. Although our understanding of the biology of HIV-1 is far from complete, the knowledge gained thus far has already led to novel strategies for both virus intervention and exploiting the lentiviruses for therapeutic applications. Reprinted by permission of the publisher.

20/3,AB/41 (Item 2 from file: 98)
DIALOG(R)File 98:General Sci Abs/Full-Text

03297530 H.W. WILSON RECORD NUMBER: BGSI96047530
Mutations in the hepatocyte nuclear factor-1a gene in maturity-onset diabetes of the young (MODY3).
Yamagata, Kazuya
Oda, Naohisa; Kaisaki, Pamela J
Nature (Nature) v. 384 (Dec. 5 '96) p. 455-8
DOCUMENT TYPE: Feature Article
SPECIAL FEATURES: bibl il ISSN: 0028-0836
LANGUAGE: English
COUNTRY OF PUBLICATION: United Kingdom

ABSTRACT: The genetic basis of maturity-onset diabetes of the young (MODY) was investigated. Two to 5 percent of cases of non-insulin-dependent (type 2) diabetes mellitus (NIDDM) are due to the single gene disorder MODY, which is characterized by autosomal dominant inheritance and an age onset of 25 years or younger. *Mutations"** in the *gene"** *TCF1"**, which encodes the transcription factor hepatocyte nuclear factor 1a (HNF-1a), were detected in subjects with the MODY3 form of NIDDM. HNF-1a plays a role in the tissue-specific regulation of the expression of several liver genes and acts as a weak transactivator of the rat insulin I gene.

20/3,AB/42 (Item 1 from file: 50)
DIALOG(R)File 50:CAB Abstracts

03333279 CAB Accession Number: 970101454
Genomic organization and chromosomal localization of the mouse Bp3 *gene"**, a member of the CD38/ADP-ribosyl cyclase family.
Dong Cheng; Willerford, D.; Alt, F. W.; Cooper, M. D.

University of Alabama at Birmingham, 378 WTI, 1824 6th Avenue South, Birmingham, AL 35294, USA.
Immunogenetics vol. 45 (1): p.35-43
Publication Year: 1996
ISSN: 0093-7711 --
Language: English
Document Type: Journal article
The mouse Bp3 antigen is expressed in early B and T lineage cells and in a discrete subpopulation of reticular cells in peripheral lymphoid tissues. The Bp3 *gene"** was cloned from a mouse genomic library and sequenced. The *gene"** consisted of 9 *exons"** and spanned approximately 27 kb. A linkage study using species-specific restriction fragment length *polymorphisms"** for Bp3 in (C57BL/6EJi x SPRET/Ei) x SPRET/Ei mice localized Bp3 near the *gene"** for CD38 on chromosome 5. The major transcriptional start site of Bp3 was mapped to -17 from the ATG start codon and was found to contain a weak initiator sequence. The upstream region lacked a TATA box, but contained consensus recognition sequences for the PU.1, Ikaros/LyF-*1"**, E2A and *TCF"**-*1"** transcription factors. Consensus motifs for cytokine responsive factors NF-IL6/C-EBP, H-APF-1/APRF and AP-1 were also present in the flanking region. Treatment with interleukin 6 increased Bp3 expression in a myeloblastoid cell line. 75 ref.

20/3,AB/43 (Item 1 from file: 442)
DIALOG(R)File 442:AMA Journals

00117268

Induction of Tyrosinase Gene Transcription in Human Iris Organ Cultures Exposed to Latanoprost (ARTICLE)

LINDSEY, JAMES D.; JONES, HEATHER L.; HEWITT, ELEANORE G.; ANGERT, MILA; WEINREB, ROBERT N.
Archives of Ophthalmology
JUNE, 2001; Laboratory: tzh853
LINE COUNT: 00653

Objective: Topical administration of latanoprost sometimes induces gradual iris darkening. The present study was undertaken to determine if latanoprost can increase transcription of the gene for tyrosinase, an important enzyme in the biosynthesis of melanin. Results from brown, hazel, and blue irides were compared. Methods: Iris tissue was isolated from 30 pairs of postmortem human donor eyes, and 2 iris segments from each eye were incubated in tissue culture medium supplemented with 200nM latanoprost acid or vehicle for 7 days. Tyrosinase messenger RNA (mRNA) was determined using real-time polymerase chain reaction analysis (TaqMan quantitative polymerase chain reaction). Results for tyrosinase mRNA were normalized according to glyceraldehyde-3-phosphate dehydrogenase (GAPDH) mRNA in each sample. Results: Tyrosinase mRNA expression was similar in blue and hazel irides, and ranged from 0.7-fold to 12.6-fold greater than GAPDH expression. In contrast, control brown iris culture tyrosinase expression ranged from6.4-fold to 265-fold greater than GAPDH expression. Induction of tyrosinase mRNA by latanoprost was below threshold in all the blue iris cultures (n=8 pairs), present in 1 of 9 hazel iris cultures, and present in 5 of 13 brown iris cultures. Mean induction in the responding hazel iris

cultures was 1.40-fold. Mean induction among the responding brown iris cultures was 2.97-fold. Conclusions: These observations support the view that iris darkening associated with latanoprost treatment reflects induction of tyrosinase expression. Moreover, they suggest that the probability that latanoprost will increase tyrosinase expression is directly related to the magnitude of tyrosinase expression before treatments are initiated. Clinical Relevance: The variability of iris darkening with latanoprost may reflect natural variation in the basal transcription of tyrosinase. Arch Ophthalmol. 2001;119:853-860

20/3,AB/44 (Item 1 from file: 65)
DIALOG(R)File 65:Inside Conferences

01668286 INSIDE CONFERENCE ITEM ID: CN017004880
Specific arrest in morphogenesis by an additional *mutation"** closely linked to two independently introduced *mutations"** in the *Tcf"**-*1"** *gene"** on mouse chromosome 11
Hofhuis, F.; Van de Wetering, M.; Clevers, H.; Verbeek, S.
CONFERENCE: Mouse molecular genetics-Meeting
ABSTRACTS OF PAPERS PRESENTED AT THE MEETING ON MOUSE MOLECULAR GENETICS , 1996 P: 102
Cold Spring Harbor Laboratory, 1996
LANGUAGE: English DOCUMENT TYPE: Conference Abstracts and programme
CONFERENCE SPONSOR: Cold Spring Harbor Laboratory (CSH)
CONFERENCE LOCATION: Cold Spring Harbor, NY
CONFERENCE DATE: Aug 1996 (199608) (199608)

Set	Items	Description
S21	546	AU=(BEGOVICH, A? OR BEGOVICH A?)
S22	2300	AU=(ERLICH, H? OR ERLICH H?)
S23	639	AU=(GRUPE, A? OR GRUPE A?)
S24	4027	AU=(NOBLE, J? OR NOBLE J?)
S25	435	AU=(PELTZ, G? OR PELTZ G?)
S26	8029	AU=(REYNOLDS, R? OR REYNOLDS R?)
S27	6119	AU=(WALKER, K? OR WALKER K?)
S28	27	AU=(ZANGENBERG, G? OR ZANGENBERG G?)
S29	0	S21 AND S22 AND S23 AND S24 AND S25 AND S26 AND S27 AND S28
S30	118	AU=(GRUPPE, A? OR GRUPPE A?)
S31	2	S21 AND S22 AND (S23 OR S30) AND S24 AND S25 AND S26 AND S27 AND S28
S32	234	S21 AND (S22 OR S23 OR S30 OR S24 OR S25 OR S26 OR S27 OR S28)
S33	181	S22 AND (S23 OR S30 OR S24 OR S25 OR S26 OR S27 OR S28)
S34	52	(S23 OR S30) AND (S24 OR S25 OR S26 OR S27 OR S28)
S35	8	S24 AND (S25 OR S26 OR S27 OR S28)
S36	5	S25 AND (S26 OR S27 OR S28)
S37	25	S26 AND (S27 OR S28)
S38	2	S27 AND S28
S39	4	(S21 OR S22 OR S23 OR S30 OR S24 OR S25 OR S26 OR S27 OR S28 OR S32 OR S33 OR S34) AND S1
S40	27	(S31 OR S35 OR S36 OR S37 OR S38 OR S39) NOT S19
S41	14	RD (unique items)

- Author(s)

>>>No matching display code(s) found in file(s): 65, 135, 453

41/3,AB/1 (Item 1 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

12169271 GENUINE ARTICLE#: 374WY NUMBER OF REFERENCES: 33
TITLE: Detection of sequence variation in the HVII region of the human mitochondrial genome in 689 individuals using immobilized sequence-specific oligonucleotide probes
AUTHOR(S): *Reynolds R (REPRINT)"**; *Walker K"**; Varlaro J; Allen M; Clark E; Alavaren M; Erlich H
CORPORATE SOURCE: Roche Mol Syst Inc, Dept Human Genet, 1145 Atlantic Ave/Alameda//CA/94501 (REPRINT); Roche Mol Syst Inc, Dept Human Genet, /Alameda//CA/94501; Univ Uppsala, Dept Genet & Pathol, /S-75105 Uppsala//Sweden/
PUBLICATION TYPE: JOURNAL
PUBLICATION: JOURNAL OF FORENSIC SCIENCES, 2000, V45, N6 (NOV), P1210-1231
PUBLISHER: AMER SOC TESTING MATERIALS, 100 BARR HARBOR DR, W CONSHOHOCKEN, PA 19428-2959 USA
ISSN: 0022-1198
LANGUAGE: English DOCUMENT TYPE: ARTICLE
ABSTRACT: We have developed a rapid, immobilized probe-based assay for the detection of sequence variation in the hypervariable segment II (HVII) of the mitochondrial DNA (mtDNA) control region. Using a panel of 17 sequence-specific oligonucleotide (SSO) probes immobilized on nylon membrane strips, we typed 689 individuals from four population groups. The genetic diversity value for each population was calculated from the frequency data, and the frequencies of distinct "mitotypes" in each group were determined. We performed DNA sequence analysis of 129 samples to characterize the sequences associated with "blanks" (absence of probe signals) and weak probe signals. Out of 689 samples, we observed five heteroplasmic samples (excluding the variable C-stretch beginning at position 303) using the immobilized SSO probe panel. The SSO probe strips were used for the analysis of shed hairs and bloodstains from several criminal cases in Sweden, one of which is described here. We conclude that this mtDNA typing system is useful for human identification and significantly decreases casework turnaround time.

41/3,AB/2 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

10133867 Genuine Article#: 483RD Number of References: 0
Title: Inheritance of specific haplotypes of the IL4R gene associated with type 1 diabetes in the HBDI families.
Author(s): Mirel DB; Valdes AM; *Reynolds RL"**; Erlich HA; *Noble JA"**
Corporate Source: Roche Mol Syst,Alameda//CA/; Childrens Hosp,Oakland Res Inst,Oakland//CA/94609
Journal: AMERICAN JOURNAL OF HUMAN GENETICS, 2001, V69, N4,1 (OCT), P 567-567
ISSN: 0002-9297 Publication date: 20011000
Publisher: UNIV CHICAGO PRESS, 1427 E 60TH ST, CHICAGO, IL 60637-2954 USA
Language: English Document Type: MEETING ABSTRACT

41/3,AB/3 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

10131856 Genuine Article#: 483RD Number of References: 0
Title: A polymorphism in the TCF7 locus is associated with type I diabetes in Caucasians.
Author(s): *Noble JA"**; White A; Mirel DB; Valdes AM; *Reynolds R"**; *Zangenberg G"**; Lazzeroni L; Grupe A; *Peltz G"**; Erlich HA
Corporate Source: Childrens Hosp,Oakland Res Inst,Oakland//CA/94609; Roche Mol Syst,Alameda//CA/; Incyte Genom,Cambridge//England/; Stanford Univ,Stanford//CA/94305; Roche Biosci,Palo Alto//CA/
Journal: AMERICAN JOURNAL OF HUMAN GENETICS, 2001, V69, N4,1 (OCT), P 226-226
ISSN: 0002-9297 Publication date: 20011000
Publisher: UNIV CHICAGO PRESS, 1427 E 60TH ST, CHICAGO, IL 60637-2954 USA
Language: English Document Type: MEETING ABSTRACT

41/3,AB/4 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

09004999 Genuine Article#: 340ZE Number of References: 0
Title: Genotyping 50 candidate SNPs simultaneously using immobilized SSO probes.
Author(s): *Reynolds R"**; *Walker K"**; Steiner L; Mirel D
Corporate Source: ROCHE MOL SYST,/ALAMEDA//CA/94501
Journal: AMERICAN JOURNAL OF MEDICAL GENETICS, 2000, V96, N4 (AUG 7), P O85-O85
ISSN: 0148-7299 Publication date: 20000807
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012
Language: English Document Type: MEETING ABSTRACT

41/3,AB/5 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13481263 BIOSIS NO.: 200200110084
Nucleotide sequence variation in the abo glycosyltransferase gene.
AUTHOR: *Reynolds R L"**; *Zangenberg G A"**
AUTHOR ADDRESS: Alameda, Calif.**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 121 (2):p1636-1637 June 9, 1998
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Citation
LANGUAGE: English
1998

41/3,AB/6 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13311134 BIOSIS NO.: 200100518283
A candidate gene approach identifies a T-cell-specific transcription factor as a possible susceptibility marker for multiple sclerosis.
AUTHOR: *Begovich A B"**(a); Grams S(a); Barcellos L F; *Reynolds R"**(a); *Walker K"**(a); Steiner L L(a); Oksenberg J R

AUTHOR ADDRESS: (a)Department of Human Genetics, Roche Molecular Systems, Alameda, CA**USA
JOURNAL: Human Immunology 62 (Supplement 1):pS29 2001
MEDIUM: print
CONFERENCE/MEETING: 27th Annual Meeting of the American Society for Histocompatibility and Immunogenetics San Francisco, California, USA October 13-17, 2001
ISSN: 0198-8859
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English
2001

41/3,AB/7 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13310730 BIOSIS NO.: 200100517879
Association of a SNP in the CTLA-4 gene with type I diabetes in Filipinos.
AUTHOR: Bugawan T L(a); Alejandrino M; Ching J(a); Panelo A; Solfelix C M; Petrone A; Buzzetti R; Pozzilli P; Steiner L(a); *Walker K"**(a); Mirell D(a); *Reynolds R"**(a); Klitz W; Erlich H A(a
AUTHOR ADDRESS: (a)Department of Human Genetics, Roche Molecular Systems, Inc., Alameda, CA**USA
JOURNAL: Human Immunology 62 (Supplement 1):pS20 2001
MEDIUM: print
CONFERENCE/MEETING: 27th Annual Meeting of the American Society for Histocompatibility and Immunogenetics San Francisco, California, USA October 13-17, 2001
ISSN: 0198-8859
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English
2001

41/3,AB/8 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13309736 BIOSIS NO.: 200100516885
Simultaneous detection of 50 candidate polymorphisms in 34 genes using megaplex PCR and linear probe arrays.
AUTHOR: *Walker K"**(a); Steiner L(a); Mirel D(a); *Reynolds R"**(a
AUTHOR ADDRESS: (a)Department of Human Genetics, Roche Molecular Systems, Alameda, CA**USA
JOURNAL: Human Immunology 62 (Supplement 1):pS145 2001
MEDIUM: print
CONFERENCE/MEETING: 27th Annual Meeting of the American Society for Histocompatibility and Immunogenetics San Francisco, California, USA October 13-17, 2001
ISSN: 0198-8859
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English
2001

41/3,AB/9 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

131195018 CA: 131(15)195018r CONFERENCE PROCEEDING
Multiplex PCR: optimization guidelines
AUTHOR(S): Zangenberg, G.; Saiki, R. K.; Reynolds, R.
LOCATION: Roche Molecular Systems, Alameda, CA, 94501, USA
JOURNAL: PCR Appl. EDITOR: Innis, Micheal A. (Ed), Gelfand, David H. (Ed), Sninsky, John J (Ed), DATE: 1999 PAGES: 73-94 CODEN: 67UAAL
LANGUAGE: English PUBLISHER: Academic,San Diego, Calif

41/3,AB/10 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

129037211 CA: 129(4)37211c PATENT
Human ABO blood group glycosyltransferase sequence polymorphism and genotyping with oligonucleotide probes
INVENTOR(AUTHOR): Reynolds, Rebecca Lynne; Zangenberg, Gabriele Annemarie
LOCATION: USA
ASSIGNEE: Roche Molecular Systems, Inc.
PATENT: United States ; US 5763184 A DATE: 19980609
APPLICATION: US 763502 (19961211)
PAGES: 15 pp. CODEN: USXXAM LANGUAGE: English CLASS: 435006000; C12Q-001/68A; C12N-015/00B; C07H-021/04B

41/3,AB/11 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

73077929 CA: 73(16)77929z JOURNAL
Examination of oil-modified alkyds and urethanes by nuclear magnetic resonance spectroscopy
AUTHOR(S): Reynolds, Reginald J. W.; Walker, Kenneth R.; Kirby, Gordon W.
LOCATION: Univ. Technol., Loughborough, Engl.
JOURNAL: Polymer DATE: 1970 VOLUME: 11 NUMBER: 6 PAGES: 333-5
CODEN: POLMAG LANGUAGE: English

41/3,AB/12 (Item 1 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS

00854802
Abo glycosyltransferase sequence polymorphism
ABO glycosyltransferase Sequenz Polymorphismus
Polymorphisme ABO en la sequence glycosyltransferase
PATENT ASSIGNEE:
F. HOFFMANN-LA ROCHE AG, (1107064), 124 Grenzacherstrasse, 4070 Basel, (CH), (applicant designated states: DE;FR;IT)
INVENTOR:
*Reynolds, Rebecca Lynne"**, 913 Independence, Alameda, California 94501, (US)
*Zangenberg, Gabriele Annemarie"**, 1440 Sixth Street, Apt. No. 14,

Alameda, California 94501, (US
LEGAL REPRESENTATIVE:
Wachter, Dieter Ernst, Dr. et al (69851), F.Hoffmann-La Roche AG Patent Department (PLP), 124 Grenzacherstrasse, 4070 Basel, (CH)
PATENT (CC, No, Kind, Date): EP 787806 A2 970806 (Basic)
APPLICATION (CC, No, Date): EP 97100830 970121;
PRIORITY (CC, No, Date): US 17117 P 960130
DESIGNATED STATES: DE; FR; IT
INTERNATIONAL PATENT CLASS: C12Q-001/68;
ABSTRACT EP 787806 A2

Methods and reagents for determining an individual's genotype at the ABO locus with respect to newly discovered polymorphisms are provided. These methods and reagents facilitate typing tissue for determining individual identity and has application in the field of forensic science. The novel reagents are oligonucleotides comprising a nucleotide sequence at least about 10 nucleotides in length, which is contained in a specified nucleotide sequence enompassing the newly discovered polymorphic site, or a complement thereof. Kits for determining the said genotypes are also provided.

ABSTRACT WORD COUNT: 84

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

Available Text	Language	Update	Word Count
CLAIMS A	(English)	9708W1	391
SPEC A	(English)	9708W1	10362
Total word count - document A			10753
Total word count - document B			0
Total word count - documents A + B			10753

41/3,AB/13 (Item 1 from file: 351)
DIALOG(R)File 351:Derwent WPI

011417448
WPI Acc No: 1997-395355/199737
XRAM Acc No: C97-127123
Oligonucleotides for detecting polymorphisms in the ABO glycosyltransferase gene - and related vectors, used forensically to identify individuals, allowing subdivision of O and B alleles
Patent Assignee: HOFFMANN LA ROCHE & CO AG F (HOFF); ROCHE MOLECULAR SYSTEMS INC (HOFF)
Inventor: *REYNOLDS R L"**; *ZANGENBERG G A"**
Number of Countries: 005 Number of Patents: 003
Patent Family:

Patent No	Kind	Date	Applicat No	Kind	Date	Week	
EP 787806	A2	19970806	EP 97100830	A	19970121	199737	B
JP 9224683	A	19970902	JP 9716318	A	19970130	199745	
US 5763184	A	19980609	US 9617117	A	19960130	199830	
			US 96763502	A	19961211		

Priority Applications (No Type Date): US 9617117 P 19960130; US 96763502 A 19961211
Patent Details:

Patent No	Kind	Lan	Pg	Main IPC	Filing Notes
EP 787806	A2	E	21	C12Q-001/68	

Designated States (Regional): DE FR IT

JP 9224683 A 19 C12N-015/09
US 5763184 A C12Q-001/68 Provisional application US 9617117

Abstract (Basic): EP 787806 A

An oligonucleotide (ON1) contains a sequence of at least 10 nucleotides (nt) from the sequence CTCCATRTGR YCGCACGCCT CTCTCCATGT GCAGTA, which represents nt 22-58 of a 161 bp sequence (I) given in the specification. Also new are: (1) oligonucleotides (ON2) that is substantially or exactly complementary to either strand of (I) in a region including a polymorphic site at nt 29, 32 or 33; where the oligonucleotide is exactly complementary at this site; and (2) DNA vectors containing ON1 or ON2.

USE - (I) is a fragment of the ABO glycosyltransferase gene and determination of R (A or G) and Y (C or T) is used to identify alleles of this gene.

Particularly ON2 are used to detect polymorphisms by hybridisation, optionally after amplification using ON2 also as the primers (so amplification and hybridisation only occur in the event of a perfect match). ON1 can be used as a positive control in the tests.The method is especially used to identify individuals for forensic purposes.

ADVANTAGE - Analysis with ON2 allows the known O and B alleles to be subdivided.

Dwg.0/0

41/3,AB/14 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotechnology Abs

0216106 DBA Accession No.: 97-11227 PATENT
Oligonucleotides for detecting polymorphisms in the ABO glycosyltransferase gene - for use in forensics
AUTHOR: *Reynolds R L"**; *Zangenberg G A"**
CORPORATE SOURCE: Basle, Switzerland.
PATENT ASSIGNEE: Roche 1997
PATENT NUMBER: EP 787806 PATENT DATE: 970806 WPI ACCESSION NO.: 97-395355 (9737)
PRIORITY APPLIC. NO.: US 17117 APPLIC. DATE: 960130
NATIONAL APPLIC. NO.: EP 97100830 APPLIC. DATE: 970121
LANGUAGE: English
ABSTRACT: New oligonucleotides (ON, DNA sequence specified) may be contained on a vector and used to transform a host cell. A method for identifying an allele present in a sample containing ABO glycosyltransferase (GT) gene nucleic acid (NA) involves determining a nucleotide base pair present at a polymorphic site. The NA is amplified and mixed with the ON under conditions wherein the ON binds to the NA to form a stable hybrid duplex only if the NA contains the variant sequence. The presence of hybrids is then detected using the bound ON as a DNA primer in an amplification reaction. This method is especially used to identify individuals for forensic purposes. In an example, a region of the ABO GT gene was amplified in a polymerase chain reaction. O alleles produce a 160 bp product and A and B alleles a 161 bp product. These products were denatured, transferred to a nylon membrane and tested for hybridization with biotinylated DNA probes for 25-30 min at 55 deg. Bound probe was detected with peroxidase (EC-1.11.1.7)/streptavidin conjugate and color formation from tetramethylbenzidine. (21pp)
? log y